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Estradiol hormone and oxidative stress markers in women with polycystic ovary syndrome

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Abstract: Polycystic ovary syndrome (PCOS) is a multifaceted metabolic and endocrine disorder affecting the female reproductive stage, characterized by infertility, anovulation, polycystic ovaries, insulin resistance, and obesity. Neuroendocrine alterations, genetics, lifestyle, and environmental pollutants are among the danger issues that contribute to PCOS in women. Hence, this study aimed to evaluate estradiol hormone (E2 and explore the grade of oxidative stress status in PCOS patients by measuring oxidant/antioxidant markers (Malondialdehyde MDA, peroxynitrite $ONOO^-$, and Glutathione GSH) in their sera. The current study included 96 women consists of 40 healthy volunteers, fertile women without PCOS, as the control group, and 56 PCOS patients who were divided into subcategories according to their BMI and age. In this study, a significant elevation of estradiol E2 and oxidant markers MDA and ONOO-) was observed among PCOS patients in contrast with non-PCOS, whereas the antioxidant marker GSH was decreased in PCOS patients. The effect of BMI within PCOS patients showed a significant elevation of E2 and oxidant biomarkers in obese PCOS, while GSH showed a significant decline as compared to non-obese PCOS. The outcomes show that the levels of E2, MDA, and ONOO- are significantly elevated with increasing age; however, GSH significantly drops.

Introduction

Polycystic ovary syndrome (PCOS) is a predominant fertility complaint [1, 2] affecting about 10.0% of middle-aged females internationally [3]. Meanwhile its first reported by Stein and Leventhal in 1935, and PCOS was identified as Stein-Leventhal syndrome [4]. Diagnosis is directed according to the Rotterdam standards: hyperandrogenism, oligoanovulation, and polycystic ovaries [5, 6]. Symptoms may be metabolic (i.e., obesity, type 2 diabetes, cardiovascular illness, dyslipidemia, hypertension, and sleep apnea), reproductive (irregular menstrual cycles, infertility, endometrial cancer, and gravidity problems), psychological (anxiety, depression, eating disorders, and poor quality of life), and dermatological (hirsutism and acne) [6-8]. Cumulative evidence proposes that oxidative stress (OS) contributes to its reproductive and metabolic complications [9].

Estradiol-E2 is a gender steroid hormone that plays an important role in male and female fertility and fitness. It is mainly formed by the female's ovary; however, minor quantities are similarly manufactured in the adrenal glands and, throughout pregnancy, via the placenta. In males, estradiol is made in lesser amounts by the testes

and adrenal glands [10]. There are four different types of estrogen: estrone (E1), estradiol (E2), estriol (E3), and estetrol (E4). Estradiol (E2) is the most powerful and abundant estrogen throughout a woman's fertile years [11]. They play a dominant part in many biological developments, mainly in the female fertility system [12]. It is vital for modifying the menstrual cycle, promoting fertility system improvement, and supporting cardiovascular and bone health [13].

Imbalance between oxidants and antioxidants is related to the production of extreme quantities of reactive oxygen species (ROS) and the ability of antioxidants in the body to protect against the harmful effects of ROS [14]. Too much ROS causes harm to DNA, lipids, and proteins, contributing to loss of cellular functions [15]. This may lead to abnormal conditions in the reproductive system, including endometriosis, preeclampsia, infertility, and recurrent abortion [16, 17]. OS plays an essential part in the pathogenesis of long-lasting illnesses such as cancer, cardiovascular diseases, diabetes, and neurodegenerative illnesses. Long exposure to high concentrations of pro-oxidant factors may lead to organizational abnormality at a mitochondrial DNA level, in addition to functional modification of numerous cellular structures and enzymes, leading to anomalies in gene expression [18, 19]. OS and Inflammation have been connected to the pathological disorder of PCOS [1].

Materials and methods

This study involves 96 women of reproductive age (21 - 40 years). 56 women with PCOS and forty same ages non PCOS, as the control group, with normal ovulating cycles (no history of infertility or hormone disorders), contributed to this work. Females with additional fertility complaints affecting productivity, like Cushing's syndrome, diabetes mellitus, hyperprolactinemia, and thyroid disease, in addition to pregnant women, were excluded from this study. PCOS female separated according to body mass index (BMI): obese PCOS females had a BMI $\geq$ 25 kg/m^2, and non-obese PCOS females had a BMI < 25 kg/m^2. BMI is calculated by the formula [20]. Furthermore, PCOS patients were grouped by age into two main age categories (younger fertility age = 21 - 30 years, and older fertility age = 31 - 40 years). Disposable syringes and needles were used for collection approximately five millimeters of venous blood from all women in early follicular phase from menstrual cycle (two or three days from the onset menstruation), immediately transferred into a non-heparinized gel separator tube, then incubated at 37 °C for ten minutes to form a coagulation in the sample before being centrifuged for 15 min at 3000 rpm to ensure separation of blood serum. The separated blood sera were used for assessments of estradiol hormone and oxidative stress markers (MDA, $ONOO^-$, and GSH). Estradiol-E_2 level was assessed using ELFA (enzyme-linked fluorescence assay). The level of malondialdehyde was estimated by an improved method described by Muslih RK and others [21], using the thiobarbituric acid (TBA) method to quantify malondialdehyde (MDA). VanuffeleneJ, et al. [23] established an altered method for assessing $ONOO^-$ concentration [22]. Finally, GSH concentration levels in sera were checked using the dithiobisnitrobenzoic acid (DTNB) method.

Ethical approval: The reference code for the approval is PREC-26-1-52. Date: 6, 2026. The ethical committee of the University of Mosul, College of Pharmacy, Mosul, Iraq (pharmacy.ethics@uomosul.edu.iq**)**.

Statistical analysis: A statistical t-test was used for analysis the data in this study. The results were expressed as means with standard deviations (SD). A significance level of $p \leq 0.05$ [24].

Results

In this study, the compares of the mean values of estradiol and oxidative stress markers between women with and without PCOS as presented in **Table 1**, the outcomes revealed that a significantly elevation of sera E2, MDA, and $ONOO^-$ levels associated with PCOS (60.09 ±0 8.77), (7.11 ± 1.43) and (69.7 ± 14.1) respectively compared to

women without PCOS (54.90 ± 10.2), (6.42 ± 0.974) and (60.9 ± 19.0) respectively. Conversely, a significant reduction of GSH in PCOS women (5.815 ± 0.985) in contrast to non-PCOS (6.41 ± 1.24) was observed.

According to the effect of BMI on estradiol and oxidative stress markers within PCOS group; as exposed in **Table 2**; the E2 and MDA, $ONOO^-$ levels showed a significant higher in obese PCOS (62.68 ± 8.03), (7.53 ± 1.18), and (73.8 ± 11.6) respectively than those of non-obese PCOS (56.64 ± 8.67), (6.56 ± 1.57), and (64.3 ± 15.3) respectively whereas GSH showed a significant reduction (5.528 ± 0.957) in comparable with non-obese PCOS (6.199 ± 0.904). **Table 3** established the effect of age on estradiol and oxidative stress status within PCOS women. The results showed that the levels of E2, MDA, and $ONOO^-$ are significantly elevated with increasing age; however, the antioxidant GSH decreased significantly.

Table 1: Estradiol and oxidative stress markers in PCOS and control groups

Parameters	Mean ± S.D.		t-value	P value
	Control group (n = 40)	PCOS group (n = 56)		
Estradiol (pg/ml)	54.90 ± 10.2	60.09 ± 8.77	- 2.67	0.0089
MDA (µmol/L)	6.42 ± 0.974	7.11 ± 1.43	- 2.65	0.0093
$ONOO^-$ (µmol/L)	60.9 ± 19.0	69.7 ± 14.1	- 2.62	0.01
GSH (µmol/L)	6.41 ± 1.24	5.815 ± 0.985	2.62	0.01

Table 2: Effect of BMI on estradiol and oxidative stress markers in the PCOS group

Parameters	Mean ± S.D.		t-value	P value
	Obese PCOS (n = 32)	Non PCOS (n = 24)		
Estradiol (pg/ml)	62.68 ± 8.03	56.64 ± 8.67	2.69	0.0095
MDA (µmol/L)	7.53 ± 1.18	6.56 ± 1.57	2.66	0.01
$ONOO^-$ (µmol/L)	73.8 ± 11.6	64.3 ± 15.3	2.66	0.01
GSH (µmol/L)	5.528 ± 0.957	6.199 ± 0.904	- 2.66	0.01

Table 3: Effect of age on estradiol and oxidative stress markers in the PCOS group

Parameters	Mean ± S.D.		t-value	P value
	Younger PCOS (n = 27)	Elderly PCOS (n = 29)		
Estradiol (pg/ml)	57.07 ± 9.04	62.91 ± 7.61	- 2.62	0.011
MDA (µmol/L)	6.62 ± 1.50	7.57 ± 1.22	- 2.59	0.012
$ONOO^-$ (µmol/L)	65.0 ± 14.6	74.1 ± 12.2	- 2.52	0.015
GSH (µmol/L)	6.159 ± 0.964	5.495 ± 0.908	2.66	0.010

Discussion

The present work focused on assessing blood serum estradiol (E2 and oxidative stress markers in female have and without PCOS, which revealed a significant elevation of E2, MDA, and ONOO- in PCOS women, while GSH showed a significant decline. MDA is a biomarker of OS, an end product of lipid peroxidation. It is a highly reactive product formed by the oxidation of polyunsaturated fatty acids (PUFAs) [25]. Peroxynitrite (ONOO-) is one of the most powerful biological nitrosative causes and has been implicated in various pathologies; it is formed when nitric oxide and superoxide combine [26]. OS state is mainly triggered by both internal metabolic processes and external lifestyle, environmental, and toxic metals exposures [27, 28]. Mitochondrial dysfunction, enzymatic

oxidative systems, and inflammatory cell engagement are the key biological causes of ROS in PCOS, and they all contribute to the growth of oxidative injury in numerous tissue compartments [29]. These findings are consistent with Sabuncu T, et al. [30], Almasoodi M. [31], and Callard and others [32], which prove that E2 is a key that encourages the growth of the endometrium and prepares it for the embedding of the embryo. If E2 levels are elevated in females with PCOS, these levels may be unbalanced, leading to irregular growth of the endometrium and an increased risk of endometrial hyperplasia, which is consistent with our results that showed raised E2 levels in PCOS women. PCOS is characterized by hyperandrogenism, and visceral and hepatic adipose tissue are the sites of additional gonadal aromatization of sex hormones caused by the activity of aromatase. This enzyme catalyzes the conversion of androgens to estrogens [33]. One of the chief mechanisms producing OS in PCOS is mitochondrial dysfunction and decreased ATP synthesis [34]. Likewise, a high androgen concentration boosts the manufacture of ROS, which deteriorates mitochondrial damage and additional oxidative injury [35, 36]. In PCOS, estrogens, particularly estradiol, have antioxidant and anti-inflammatory properties [37]. E2, which owns a phenolic hydroxyl group, has real antioxidant activity and prevents lipid peroxidation in model membranes. Antioxidant properties of E2 have been proposed for the prevention of oxidation of LDL, an important phase in atherogenesis [38-41]. Furthermore, GSH is a non-enzymatic antioxidant that acts to detoxify H2O2 in human cells by giving an electron and is changed to the oxidized form (GSSG) [42]. The effect of BMI and age in women with PCOS highlights a significant elevation of serum estradiol, MDA, and ONOO- in obese women with PCOS compared to non-obese women with PCOS, while GSH was decreased. Similar results were observed in elderly PCOS women compared with younger PCOS women. These findings are in agreement with Uçkan K, et al. [43] and Fatima S, et al. [44].

Conclusion: Oxidative stress has detrimentally influence on human health, which is widely recognized as a central player in the pathophysiology of PCOS. It creates a state of imbalance between harmful ROS creation and the body's ability to counteract them, which directly disrupts ovarian function. Moreover, BMI and age play a crucial role in oxidative stress status and contribute to it within PCOS.

References

1. Su P, Chen C, Sun Y. Physiopathology of polycystic ovary syndrome in endocrinology, metabolism and inflammation. Journal of Ovarian Research. 2025; 18(1): 34. doi: 10.1186/s13048-025-01621-6
2. Escalona XA, Ayala RS, Cortez K, Sánchez SF, Tome-Dehesa T, Díaz-Hernández V, et al. Comparative analysis of letrozole and estradiol valerate PCOS models: Reproductive and metabolic outcomes with and without high-fat diet. Biology 2025; 14: 592. doi: 10.3390/ biology14060592
3. Zhao W, Zhou J, Song Y, He M, Zhu X, Yu B, Gao W. Polycystic ovary syndrome revisited: Novel insights and updates. International Journal of Medical Sciences. 2026; 23(1): 271-282. doi: 10.7150/ijms.119968
4. Stein IF, Leventhal ML. Amenorrhea associated with bilateral polycystic ovaries. American Journal of Obstetrics and Gynecology. 1935; 29(2): 181-191. doi: 10.1016/S0002-9378(15)30642-6
5. Ghafari A, Maftoohi M, Samarin M E, Barani S, Banimohammad M, Samie R. The last update on polycystic ovary syndrome (PCOS), diagnostic criteria, and novel treatment. Endocrine and Metabolic Science. 2025; 17(1):100228. doi: 10.1016/j.endmts.2025.100228
6. Teede HJ, Tay CT, Laven JJE, Dokras A, Moran LJ, Piltonen TT, et al, and the International PCOS Network. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. European Journal of Endocrinology. 2023; 189(2): G43-G64. doi: 10.1093/ejendo/ lvad096
7. Peña AS, Witchel SF, Boivin J, Burgert TS, Ee C, Hoeger KM, Lujan ME, et al. International evidence-based recommendations for polycystic ovary syndrome in adolescents. BMC Medicine 2025; 23(1): 151. doi: 10.1186/ s12916-025-03901-w
8. Stener-Victorin E, Teede H, Norman RJ, Legro R, Goodarzi MO, Dokras A, Laven J, Hoeger K, et al. Polycystic ovary syndrome. Nature Reviews Disease Primers. 2024; 10(1): 27. doi: 10.1038/s41572-024-00511-3

9. Zuhra H, Saifullah S, Inayat B, Mehr A, Arif A, Khalid S. Association of oxidative stress biomarkers with polycystic ovary syndrome (PCOS) and its metabolic outcomes, including insulin resistance and dyslipidemia: A systematic review: Oxidative stress biomarkers with PCOS: Insulin resistance and dyslipidemia. Pakistan Journal of Health Sciences. 2025; 6(11), 171-178. doi: 10.54393/pjhs.v6i11.3504
10. Oyant A. Estradiol: An overview of its role, functions and importance. Journal of Fertilization: In Vitro IVF Worldwide, Reproductive Medicine, Genetics and Stem Cell Biology. 2025; 13: 406. doi: 10.35841/2329-9495. 25.13.406
11. Thomas MP, Potter BV. The structural biology of oestrogen metabolism. The Journal of Steroid Biochemistry and Molecular Biology. 2013; 137: 27-49. doi: 10.1016/j.jsbmb.2012.12.014
12. Mydeen F Sangeetha K. Role of estrogen and estrogen receptor in polycystic ovarian syndrome - A molecular insight from past to present. Current Pharmacology Reports. 2025; 11(1): 47. doi: 10.1007/s40495-025-00423-0
13. Anagnostis P, Bosdou JK, Vaitsi K, Goulis DG, Lambrinoudaki I. Estrogen and bones after menopause: A reappraisal of data and future perspectives. Hormones (Athens). 2021; 20(1): 13-21. doi: 10.1007/s42000-020-00218-6
14. Li B, Ming H, Qin S, Nice EC, Dong J, Du Z, Huang C. Redox regulation: mechanisms, biology and therapeutic targets in diseases. Signal Transduction and Targeted Therapy. 2025; 10(1): 72. doi: 10.1038/s41392-024-02095-6
15. Cruikshank A, Nijhout HF, Reed MC. The role of estradiol in oxidative stress in health and cystathionine ß-synthase deficiency: A mechanistic model. CPT Pharmacometrics and Systems Pharmacology. 2026; 15(4): e70243. doi: 10.1002/psp4.70243
16. Zeber-Lubecka N, Ciebiera M, Hennig EE. Polycystic ovary syndrome and oxidative stress-from bench to bedside. International Journal of Molecular Sciences. 2023; 24: 14126. doi: 10.3390/ijms241814126
17. AL-Hamdani I H, Al-Helaly LA. Peroxiredoxin 3 and oxidative stress in recurrent abortion patients. Military Medical Sciences Letters. 2023; 92(1): 87-94. doi: 10.31482/mmsl.2022.034
18. Kıran T, Otlu O, Karabulut A. Oxidative stress and antioxidants in health and disease. Journal of Laboratory Medicine. 2023; 47(1): 1-11. doi: 10.1515/labmed-2022-0108
19. Pooja G, Shweta S, Patel P. Oxidative stress and free radicals in disease pathogenesis: A review. Discover Medicine. 2025; 2: 104. doi: 10.1007/s44337-025-00303-y
20. Haslett C, Chilvers ER, Boon NA, Colledge NR. Davidson's principles and practice of medicine. 19th ed., Churchill Livingstone: 2002; 835-837. ISBN-13: 978-0443071119
21. Muslih RK, Al-Nimer MS, Al-Zamely OY. The level of malondialdehyde after activation with H2O2 and CuSO4 and inhibition by deferoxamine and Molsidomine in the serum of a patient with acute myocardial infarction. Nature. Journal of Chemistry. 2002; 5: 139-148. doi: Nil.
22. Vanuffelen BE, Van Der Zee J, De Koster BM, Vansteveninck J, Elferink JG. Intracellular but not extracellular conversion of nitroxyl anion into nitric oxide leads to stimulation of human neutrophil migration. Biochemical Journal. 1998; 33(2): 719-722. doi: 10.1042/bj3300719
23. Sedlak J, Lindsay RH. Estimation of total, protein-bound, and nonprotein sulfhydryl groups in tissue with Ellman's reagent. Analytical Biochemistry. 1968; 25(1): 192-205. doi: 10.1016/0003-2697(68)90092-4
24. du Prel JB, Röhrig B, Blettner M. Statistical methods in medical research. Deutsches Ärzteblatt International. 2009; 106(7): 99. doi: 10.3238/arztebl.2009.0099
25. Qasim MN, Mahmood HG. Assessment of malondialdehyde as a biomarker of oxidative stress and its correlation with 25-hydroxy vitamin D3 in women with polycystic ovary syndrome oxy vitamin D3. Journal of Faculty of Medicine Baghdad. 2026; 67(3): 330-337. doi: Nil.
26. Piacenza L, Zeida A, Trujillo M, Radi R. The superoxide radical switch in the biology of nitric oxide and peroxynitrite. Physiological Reviews. 2022; 102(4): 1881-1906. doi: 10.1152/physrev.00005.2022
27. Liu S, Liu J, Wang Y, Deng F, Deng Z. Oxidative stress: Signaling pathways, biological functions, and disease. MedComm (2020). 2025; 6(7): e70268. doi: 10.1002/mco2.70268
28. AL-Hamdani I H, Al-Helaly LA. Peroxiredoxin 3 with Toxic metals in Missed Abortion Patients. Military Medical Sciences Letters. 2023; 92(3): 272-279. doi: 10.31482/mmsl.2022.050
29. Rudnicka E, Duszewska AM, Kucharski M, Tyczyński P, Smolarczyk R. Oxidative stress and reproductive function: Oxidative stress in polycystic ovary syndrome. Reproduction (Cambridge, England). 2022; 164(6): F145-154. doi: 10.1530/REP-22-0152
30. Sabuncu T, Vural H, Harma M, Harma M. Oxidative stress in polycystic ovary syndrome and its contribution to the risk of cardiovascular disease. Clinical Biochemistry. 2001; 34(5): 407-413. doi: 10.1016/s0009-9120(01)00245-4

31. Almasoodi M. Hormonal imbalance of estrogen and progesterone, its association with PCOS, and its impact on pregnancy rates in women from Thi-Qar Governorate. University of Thi-Qar Journal of Science. 2025; 12: 142-147. doi: 10.32792/utq/utjsci/v12i1.1358
32. Callard GV, Tarrant AM, Novillo A, Yacci P, Ciaccia L, Vajda S, et al. Evolutionary origins of the estrogen signaling system: Insights from amphioxus. Journal of Steroid Biochemistry and Molecular Biology. 2011; 127: 176-188. doi: 10.1016/j.jsbmb.2011.03.022
33. Chen J, Shen S, Tan Y, Xia D, Xia Y, Cao Y, et al. The correlation of aromatase activity and obesity in women with or without polycystic ovary syndrome. Journal of Ovarian Research. 2015; 8: 11. doi: 10.1186/s13048-015-0139-1
34. Gao Y, Zou Y, Wu G, Zheng L. Oxidative stress and mitochondrial dysfunction of granulosa cells in polycystic ovarian syndrome. Frontiers in Medicine (Lausanne). 2023; 10: 1193749. doi: 10.3389/fmed.2023.1193749
35. Rizk J, Sahu R, Duteil D. An overview on androgen-mediated actions in skeletal muscle and adipose tissue. Steroids. 2023; 199: 109306. doi: 10.1016/j.steroids.2023.109306
36. Wang K, Li Y, Chen Y. Androgen excess: A hallmark of polycystic ovary syndrome. Frontiers in Endocrinology. 2023; 14: 1273542. doi: 10.3389/fendo.202 3.1273542
37. Abraham DMB, Ke RW, Bloomer RJ. Impact of serum estradiol on biomarkers of oxidative stress in polycystic ovary syndrome and ovulatory women. Clinical Medicine Insights: Women's Health. 2012; 45-51. doi: 10.4137/ CMWH.S9721
38. Subbiah MT, Kessel B, Agrawal M, Rajan R, Abplanalp W, Rymaszewski Z. Antioxidant potential of specific estrogens on lipid peroxidation. Journal of Clinical Endocrinology and Metabolism. 1993; 77(4): 1095-1097. doi: 10.1210/jcem.77.4.8408459
39. Santanam N, Shern-Brewer R, McClatchey R, Castellano PZ, Murphy AA, Voelkel S, Parthasarathy S. Estradiol as an antioxidant: Incompatible with its physiological concentrations and function. Journal of Lipid Research. 1998. 39(11): 2111-2118. PMID: 9799796.
40. AL-belazi BJ, Aldiab LM, Sherif FM. Inositol has a beneficial effect in the management of polycystic ovary syndrome: Special data for Libyan patients. Mediterranean Journal of Medical Research. 2026; 3(1): 14-26. doi: 10.5281/zenodo.18203319
41. Alharati SH, Elbakay JAM, Hermann A, Gabj AM. Polycystic ovary syndrome: Molecular modeling study on potential Lepidium sativum bioactive compounds in modulating kiss-1 gene function. Mediterranean Journal of Medical Research. 2025; 2(3): 129-140. doi: 10.5281/zenodo.17069661
42. Luque-Ceballos J C, Rodríguez-Zamora P, López-Olivos J C, Garzón I L. Revisiting the scavenging activity of glutathione: Free radicals' diversity and reaction mechanisms. Computational and Theoretical Chemistry. 2023; 1227: 114227. doi: 10.1016/j.comptc.2023.114227
43. Uçkan K, Demir H, Turan K, Sarıkaya E, Demir C. Role of oxidative stress in obese and nonobese PCOS patients. International Journal of Clinical Practice. 2022; 2022: 4579831. doi: 10.1155/2022/4579831
44. Fatima S, Saeedullah A, Mahmood A, Iqbal M, Firdous S, Alim U. Oxidative stress and lipid peroxidation markers in obesity-related polycystic ovary syndrome and menstrual irregularities: A systematic review of biochemical and pharmacological interventions: Oxidative stress and lipid peroxidation markers in obesity-related PCOS. Pakistan Journal of Health Sciences. 2026; 7(2): 151-160. doi: 10.54393/pjhs.v7i2.3609

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